Check for updates

Received: 30 July 2024 | Accepted: 4 April 2025

DOI: 10.1002/jhm.70063

Journal of Hospital Medicine | shm Society of Hospital Medicine

CHOOSING WISELY®

THINGS WE DO FOR NO REASON

Things We Do for No Reason™: Avoiding intravenous iodinated contrast material in patients with acute kidney injury or chronic kidney disease

Caroline Coleman MD[1,2] | **Meredith Trubitt MD, MPH**[1,2] | **Joel Topf MD, FACP**[3] | **Monee Amin MD**[1,2]

[1]Department of Medicine, Division of Hospital Medicine, Emory University School of Medicine, Atlanta, Georgia, USA

[2]Joseph Maxwell Cleland Atlanta VA Medical Center, Decatur, Georgia, USA

[3]Division of Nephrology, Department of Medicine, Oakland University William Beaumont School of Medicine, Rochester, Michigan, USA

Correspondence

Caroline Coleman, MD, Department of Medicine, Division of Hospital Medicine, Emory University School of Medicine, Atlanta, GA, USA.
Email: Caroline.coleman@emory.edu

CLINICAL CASE

The hospitalist admits a 50-year-old man with the past medical history of chronic kidney disease (CKD) stage 3a for tachycardia, dyspnea, and unilateral leg swelling. The hospitalist has a high clinical suspicion for pulmonary embolism and wants to obtain a computed tomography (CT) chest with contrast, but she worries that the patient will develop contrast-induced acute kidney injury (CI-AKI) after receiving intravenous (IV) iodinated contrast material (ICM), aka "contrast nephropathy."

WHY HOSPITALISTS MIGHT BELIEVE THAT IV ICM CAUSES RENAL DYSFUNCTION

The literature has associated IV ICM with AKI since a 1954 case report of a patient who developed acute anuric renal failure after IV pyelography.[1] A series of well-done IV ICM randomized, but not controlled, trials in the 1990s and 2000s focused on relative toxicities of various contrast agents and found rates of AKI ranging between 2.3% and 27.8%.[2–4] Even the American College of Radiology (ACR) and National Kidney Foundation (NKF) have a definition for CI-AKI: an AKI occurring within 48 h of receipt of ICM with exclusion of other nephrotoxic factors.[5]

WHY THERE IS LITTLE REASON TO BE AFRAID OF IV ICM IN INDIVIDUALS WITH HIGH GLOMERULAR FILTRATION RATES (GFR)

Four arguments demonstrate why hospitalists do not need to avoid IV ICM in patients with AKI or CKD. First, a growing body of data suggests that receiving IV ICM may correlate with, but does not cause, worsening renal function. Second, researchers have questioned the pathophysiologic basis of CI-AKI. Third, even if ICM causes AKI, patients rarely experience patient-centered renal outcomes like dialysis. Fourth, withholding IV ICM may subject patients to diagnostic delays and worse clinical outcomes, known as "renalism."

Previously, investigators have followed the dogma that IV ICM causes AKI rather than assuming a simple correlation between the two.[2,4,6] Researchers created studies without control groups that primarily compared outcomes between low-osmolar and iso-osmolar ICM. That began to change in 2008 when a single-center retrospective study by Newhouse et al.[7] assessed the frequency and magnitude of serum creatinine changes in >30,000 hospitalized patients with serum or plasma creatinine measurements on 5 consecutive days who did not receive ICM. They found creatinine increases did not significantly differ from previously published rates seen in contrast nephropathy, suggesting that hospitalized patients experience a significant baseline rate of AKI regardless of exposure to ICM. Additionally, Brinjikji et al.'s systematic review and meta-analysis[8] analyzed patients both with and without

 © 2025 Society of Hospital Medicine

SHOUT
SHARE
IT

CKD receiving ICM as part of acute stroke workup. Researchers found a lower incidence of AKIs, odds ratio = 0.47; 95% confidence interval = 0.33–0.68; $p < .01$, in patients who received contrast compared with patients who did not. Selection bias likely explains this surprising finding. Providers may have preferentially selected patients perceived as lower risk to receive ICM, increasing the proportion of high-risk patients in the non-ICM group who might develop more AKIs, regardless of etiology. Further studies used propensity score matching to combat selection bias. A series of studies from 2014 to 2017 showed no difference in AKIs between groups that did or did not receive ICM,[9,10] even when stratified by estimated glomerular filtration rate (eGFR).[8,11] In a data set that included nearly 6,000,000 hospitalized adults with a length of stay <10 days, Wilhelm-Leen et al.[12] demonstrated a slight but significantly higher incidence of AKI in patients who did not receive ICM versus those who did (5.6% vs. 5.5%, $p = .51$; after controlling for comorbidity and acuity of illness, OR for AKI 0.93, 95% CI 0.88–0.97). They concluded that "the relation between radiocontrast administration and AKI is highly confounded, unpredictable, and sometimes bidirectional." This accumulation of evidence led the *New England Journal of Medicine* to modify its nomenclature in its review on the topic from "Preventing Nephropathy Induced by Contrast Medium" in 2006[13] to "contrast-associated AKI" in 2019.[14] The authors cited residual confounding and indication bias as major limitations of earlier studies.

Second, the basic pathophysiologic arguments for CI-AKI remain unsettled. CI-AKI results from the interplay of both cytotoxic and hypoxic pathways, but researchers have yet to fully elucidate the pathophysiology.[14] Many researchers stop short of calling the process acute tubular necrosis (ATN), due to inconsistent features such as a shorter time to renal recovery than other types of ATN (i.e., days rather than weeks).[15] Several studies[16,17] have compared increases in creatinine, a functional marker, versus renal biomarkers—clinically unavailable tests that directly reflect tissue damage to renal cells—following administration of intraarterial ICM. These studies found no rise in biomarkers, even in patients with rising creatinine levels meeting the definition of contrast-associated AKI. This suggests that hemodynamic processes unrelated to ICM led to the rise in creatinine rather than direct injury to nephrons from ICM.[16,17]

Third, patients experience very low rates of progression of CKD and initiation of dialysis following administration of IV ICM. In 2021, Goulden et al.[18] studied patients with and without CKD who had a D-dimer result just above and below the threshold of 500 ng/mL, effectively randomizing 156,000 patients to receive IV ICM during a pulmonary embolism evaluation. The analysis revealed no increased AKI, CKD at 6 months, renal replacement therapy, or mortality between groups at all eGFR stratifications, including eGFR <30 mL/min/1.73 m^2. The Kompas trial[19] randomized 523 patients with stage 3 CKD undergoing contrasted CT scans to receive no prehydration versus prehydration with sodium bicarbonate. Kompas found that no patients required dialysis within 1 year. Before undergoing an elective procedure that required ICM, the Nijssen et al.[20] randomized patients 1:1 at high risk (eGFR 30–59 mL/min/1.73 m^2) of developing CI-AKI to normal saline prophylaxis versus no prophylaxis. None of the 660 patients required dialysis within 35 days and the authors found no statistically significant difference in the rates of contrast-induced nephropathy.[20]

This evidence spurred a joint consensus statement from ACR and NKF in 2020.[5] ACR/NKF reiterated that the medical community has overstated the risk of CI-AKI and noted that hospitalists should not withhold ICM based on kidney function if required for assessment of potentially life-threatening diagnoses. Despite this body of evidence, many practitioners adopt a "better safe than sorry approach" to ICM, a phenomenon acknowledged in the 2020 ACR/NKF consensus statement. This practice pattern produces its own risk. Chertow et al.[21] first described the risk as "renalism": prioritizing perceived renal health over the health of other organs. This manifests when hospitalists avoid prescribing testing involving ICM in patients with renal dysfunction even though the testing may improve outcomes overall. The Chertow cohort study found that elderly patients with CKD (creatinine >1.5) who underwent coronary angiography with a diagnosis of myocardial infarction had a lower 1-year mortality, 29.8%, compared with those who did not undergo coronary angiography, 47.4%.[21] They asserted that "renalism" discourages the performance of important studies and leads to harm overall.

WHEN ICM MAY CAUSE CONCERN FOR RENAL DYSFUNCTION

Due to limited data, hospitalists should take a judicious approach to IV ICM administration in those with eGFR<30 mL/min/1.73 m^2.[22] In that population, the ACR/NKF consensus statement estimates a number needed to harm between 6 and infinity, demonstrating the heterogeneity of current evidence.[6] Since publication of the consensus statement, a well-done study has questioned even this recommendation. In 2023, Ehmann et al.[23] analyzed patients admitted with pre-existing AKI who subsequently received ICM. They found that IV contrast administration did not increase the risk of persistent AKI at discharge or initiation of dialysis within 180 days, including for the 38% of their cohort that presented to the ED with an eGFR <30 mL/min/1.73 m^2.

WHAT HOSPITALISTS SHOULD DO INSTEAD

CI-AKI mitigation strategies may help high-risk patients. The 2020 ACR/NKF consensus statement[5] recommended using prophylactic volume expansion with normal saline for patients not undergoing maintenance dialysis who have any degree of AKI or eGFR <30 mL/min/1.73 m^2. However, the authors note that the evidence for this recommendation comes from studies without a well-matched control group that could establish a potential causal relationship, and that hospitalists must weigh possible mitigation of AKI against the risk of hypervolemia (e.g., comorbid heart failure).[5] The consensus statement does not provide ideal timing, amount, or rate for volume expansion. The consensus statement also

15535606, 0, Downloaded from https://shmpublications.onlinelibrary.wiley.com/doi/10.1002/jhm.70063 by dr nicholasbrandon - Faculty Of Medicine Chulalongkorn University , Wiley Online Library on [21/05/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

suggests considering volume expansion prophylaxis in high-risk patients with eGFR of 30–44 mL/min/1.73 m^2. Two recent randomized control trials, however, demonstrated the futility of IV fluids in patients with CKD 3 (eGFR 30–59 mL/min/1.73 m^2).[19,20]

RECOMMENDATIONS

- To protect the patient from "renalism," acknowledge bias against administering IV ICM to patients with AKI and CKD and the resultant potential risk to other organs.
- In a life-threatening situation, do not withhold IV ICM based on kidney function.
- In routine situations, do not withhold IV ICM for patients if eGFR ≥30 mL/min/1.73 m^2.
- In routine situations, consider the risks and benefits of ICM administration if the eGFR is <30 mL/min/1.73 m^2.
- Use prophylactic isotonic volume expansion with normal saline in select patients with eGFR<30 mL/min/1.73 m^2 when benefits exceed the risk of potential hypervolemia, and consider it for those with eGFR 30–45 mL/min/1.73 m^2.

CONCLUSION

If CI-AKI represents a true clinical entity, it is rare. Elevations in serum creatinine likely represent markers—not mediators—of serious adverse outcomes. This can reassure the hospitalist that in the setting of urgent procedures, the risk of contrast is low, and, conversely, the risk of not doing a procedure may cause more harm. Renalism carries real risk for CKD and AKI patients. Nephrologists and radiologists agree that hospitalists should not withhold needed IV ICM based on kidney function.

Returning to our case, the hospitalist weighs the risks and benefits of IV ICM and opts to obtain the CT chest with contrast. Despite the hospitalist's clinical suspicion, the CT finds no pulmonary emboli. While the serum creatinine following the procedure initially rises, it improves with IV hydration and time.

ACKNOWLEDGMENTS

The authors thank Ms. Mary Ann Kirkconnell Hall, MPH for editorial assistance. The authors have no funding to report.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

ORCID

Caroline Coleman http://orcid.org/0000-0002-4035-751X

X (FORMERLY KNOWN AS TWITTER)

Meredith Trubitt 𝕏@MeredithTrubitt

Monee Amin 𝕏@monsta23

REFERENCES

1. Bartels ED, Brun GC, Gammeltoft A, GjØRup PA. Acute annria following intravenous pyelography In a patient with myelomatosis. *Acta Med Scand*. 1954;150(4):297-302. doi:10.1111/j.0954-6820.1954.tb18632.x
2. Barrett BJ, Katzberg RW, Thomsen HS, et al. Contrast-induced nephropathy in patients with chronic kidney disease undergoing computed tomography: a double-blind comparison of iodixanol and iopamidol. *Invest Radiol*. 2006;41(11):815-821. doi:10.1097/01.rli.0000242807.01818.24
3. Nguyen SA, Suranyi P, Ravenel JG, et al. Iso-osmolality versus low-osmolality iodinated contrast medium at intravenous contrast-enhanced CT: effect on kidney function. *Radiology*. 2008;248(1):97-105. doi:10.1148/radiol.2481071484
4. Thomsen HS, Morcos SK. Risk of contrast-medium-induced nephropathy in high-risk patients undergoing MDCT—a pooled analysis of two randomized trials. *Eur Radiol*. 2009;19(4):891-897. doi:10.1007/s00330-008-1206-4
5. Davenport MS, Perazella MA, Yee J, et al. Use of intravenous iodinated contrast media in patients with kidney disease: consensus statements from the American College of Radiology and The National Kidney Foundation. *Radiology*. 2020;294(3):660-668. doi:10.1148/radiol.2019192094
6. McCullough PA, Brown JR. Effects of intra-arterial and intravenous iso-osmolar contrast medium (iodixanol) on the risk of contrast-induced acute kidney injury: a meta-analysis. *Cardiorenal Med*. 2011;1(4):220-234. doi:10.1159/000332384
7. Newhouse JH, Kho D, Rao QA, Starren J. Frequency of serum creatinine changes in the absence of iodinated contrast material: implications for studies of contrast nephrotoxicity. *Am J Roentgenol*. 2008;191(2):376-382. doi:10.2214/AJR.07.3280
8. Brinjikji W, Demchuk AM, Murad MH, et al. Neurons over nephrons: systematic review and meta-analysis of contrast-induced nephropathy in patients with acute stroke. *Stroke*. 2017;48(7):1862-1868. doi:10.1161/STROKEAHA.117.016771
9. McDonald JS, McDonald RJ, Carter RE, Katzberg RW, Kallmes DF, Williamson EE. Risk of intravenous contrast material-mediated acute kidney injury: a propensity score-matched study stratified by baseline-estimated glomerular filtration rate. *Radiology*. 2014;271(1):65-73. doi:10.1148/radiol.13130775
10. McDonald RJ, McDonald JS, Bida JP, et al. Intravenous contrast material-induced nephropathy: causal or coincident phenomenon? *Radiology*. 2013;267(1):106-118. doi:10.1148/radiol.12121823
11. Hinson JS, Ehmann MR, Fine DM, et al. Risk of acute kidney injury after intravenous contrast media administration. *Ann Emerg Med*. 2017;69(5):577-586.e4. doi:10.1016/j.annemergmed.2016.11.021
12. Wilhelm-Leen E, Montez-Rath ME, Chertow G. Estimating the risk of radiocontrast-associated nephropathy. *J Am Soc Nephrol*. 2017;28(2):653-659. doi:10.1681/ASN.2016010021
13. Barrett BJ, Parfrey PS. Preventing nephropathy induced by contrast medium. *N Engl J Med*. 2006;354(4):379-386. doi:10.1056/NEJMcp050801
14. Mehran R, Dangas GD, Weisbord SD. Contrast-associated acute kidney injury. *N Engl J Med*. 2019;380(22):2146-2155. doi:10.1056/NEJMra1805256
15. Fang LST. Low fractional excretion of sodium with contrast media-induced acute renal failure. *Arch Intern Med*. 1980;140(4):531-533. doi:10.1001/archinte.1980.00330160091033
16. Liu C, Mor MK, Palevsky PM, et al. Postangiography increases in serum creatinine and biomarkers of injury and repair. *Clin J Am Soc Nephrol*. 2020;15(9):1240-1250. doi:10.2215/CJN.15931219
17. Kooiman J, van de Peppel WR, Sijpkens YWJ, et al. No increase in kidney injury molecule-1 and neutrophil gelatinase-associated lipocalin excretion following intravenous contrast enhanced-CT. *Eur Radiol*. 2015;25(7):1926-1934. doi:10.1007/s00330-015-3624-4

15535606, 0, Downloaded from https://shmpublications.onlinelibrary.wiley.com/doi/10.1002/jhm.70063 by dr nicholashrandon - Faculty Of Medicine Chulalongkorn University , Wiley Online Library on [21/05/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

18. Goulden R, Rowe BH, Abrahamowicz M, Strumpf E, Tamblyn R. Association of intravenous radiocontrast with kidney function: a regression discontinuity analysis. *JAMA Internal Medicine*. 2021;181(6):767-774. doi:10.1001/jamainternmed.2021.0916
19. Timal RJ, Kooiman J, Sijpkens YWJ, et al. Effect of no prehydration vs sodium bicarbonate prehydration prior to contrast-enhanced computed tomography in the prevention of postcontrast acute kidney injury in adults with chronic kidney disease: the Kompas randomized clinical trial. *JAMA Internal Medicine*. 2020;180(4):533-541. doi:10.1001/jamainternmed.2019.7428
20. Nijssen EC, Rennenberg RJ, Nelemans PJ, et al. Prophylactic hydration to protect renal function from intravascular iodinated contrast material in patients at high risk of contrast-induced nephropathy (AMACING): a prospective, randomised, phase 3, controlled, open-label, non-inferiority trial. *The Lancet*. 2017;389(10076):1312-1322. doi:10.1016/S0140-6736(17)30057-0
21. Chertow GM, Normand SLT, McNeil BJ. Renalism": inappropriately low rates of coronary angiography in elderly individuals with renal insufficiency. *J Am Soc Nephrol*. 2004;15(9):2462-2468. doi:10.1097/01.ASN.0000135969.33773.0B
22. Dekkers IA, van der Molen AJ. Propensity score matching as a substitute for randomized controlled trials on acute kidney injury after contrast media administration: a systematic review. *Am J Roentgenol*. 2018;211(4):822-826. doi:10.2214/ajr.17.19499
23. Ehmann MR, Mitchell J, Levin S, et al. Renal outcomes following intravenous contrast administration in patients with acute kidney injury: a multi-site retrospective propensity-adjusted analysis. *Intensive Care Med*. 2023;49(2):205-215. doi:10.1007/s00134-022-06966-w

How to cite this article: Coleman C, Trubitt M, Topf J, Amin M. Things We Do for No Reason™: Avoiding intravenous iodinated contrast material in patients with acute kidney injury or chronic kidney disease. *J Hosp Med*. 2025;1-4. doi:10.1002/jhm.70063

15535606, 0, Downloaded from https://shmpublications.onlinelibrary.wiley.com/doi/10.1002/jhm.70063 by dr nicholasbrandon - Faculty Of Medicine Chulalongkorn University , Wiley Online Library on [21/05/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License